

Only in the treatment of severe bradycardia, caused by increased vagal tone, may atropine be preferred to glycopyrrolate because of its greater effectiveness in increasing pulse rate. In all other pharmacological actions, glycopyrrolate appears to have decided advantages.

KENNETH S. CHING, MD

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## The Pulmonary Artery Catheter for Measurement of Left Ventricular Function

ASSESSMENT of left ventricular performance and reserve is important for diagnostic evaluation and treatment of critically ill patients in an intensive care unit (ICU) and during major surgical procedures. Ventricular performance is determined primarily by heart rate and cardiac contractility, preload and afterload.

The heart rate is measured easily from an electrocardiographic monitor. If diastolic filling is maintained constant, cardiac output will increase as heart rate increases. Myocardial contractility may be assessed indirectly by invasive or non-invasive methods, but a practical method for continuous monitoring of contractility is not yet available in clinical practice. Afterload is the myocardial wall tension developed during contraction and is proportional to arterial blood pressure. Therefore, it is easily assessed by conventional blood pressure monitoring.

The accurate assessment of preload, defined as myocardial fiber length at the end of diastole, presents a difficult problem. Preload is related to ventricular volume, and ventricular volume is, in turn, roughly proportional to left ventricular end-diastolic pressure (LVEDP). Although ventricular volume and LVEDP change in the same direction, the relationship is not linear in normal hearts and it is further altered by conditions which change myocardial compliance. Mean left atrial pressure (mLAP) is frequently used in the clinical setting to assess LVEDP since the two are nearly identical in patients with normal hearts, but mLAP is an inaccurate reflection of LVEDP in patients with abnormal left ventricular function, and it can only be measured by direct insertion of a

catheter through the left atrial wall at operation.

The introduction of a balloon-tipped flow-directed pulmonary artery catheter made available an effective and reasonably safe clinical method for assessment of mLAP (and, therefore, indirectly, preload).

In its simplest form, the pulmonary artery catheter has two lumina. One extends the entire length of the catheter, opening at the tip, through which it is possible to measure pressure and collect blood samples. The other lumen opens just proximal to the catheter tip and is used to inflate the balloon. When the catheter is placed in a peripheral branch of a pulmonary artery, balloon inflation will obstruct arterial blood flow in that segment of the pulmonary arterial system. The pressure then recorded beyond the balloon is no longer pulmonary artery pressure but becomes the pressure transmitted retrograde from the left atrium. This pulmonary artery occluded pressure (PAOP) is essentially identical to pulmonary wedge pressure (PWP). A close relationship exists between mLAP and PWP when PWP is less than 25 mm of mercury. Measurement of PAOP consequently allows for a relatively accurate determination of mLAP which in turn corresponds to LVEDP. As LVEDP increases so does PAOP.

Modifications of the catheter include an additional lumen which opens into the right atrium, and a thermistor probe 10 cm proximal to the tip of the catheter, for measuring cardiac output by thermodilution. The versatility of the pulmonary artery catheter is notably increased by these two additions. For example, it is also possible utilizing body surface area, heart rate, cardiac output, mean arterial pressure, mean right atrial pressure and PAOP to calculate stroke volume index, total peripheral resistance, pulmonary vascular resistance, and left and right ventricular stroke work index.

Because of the disparity between central venous pressure and PAOP in patients with compromised left ventricular function, uncompensated cirrhosis, advanced peritonitis or multisystem trauma, catheterization of the pulmonary artery is indicated for adequate monitoring of fluid therapy or a hemodynamic intervention in the ICU or during anesthesia. Other uses are in the management of patients during controlled hypotension and in monitoring massive volume replacement.

Premature ventricular contractions may occur during 17 percent of attempted catheter placements. The arrhythmias are transient, and usually

subside when the catheter tip is in the pulmonary artery. Symptomatic pulmonary infarction may result if the catheter floats into a permanent wedge position or the balloon is left inflated for a prolonged time. Thrombosis associated with the pulmonary artery catheter has been reported, as has perforation of the pulmonary artery and knotting about intracardiac structures. Balloon rupture may occur if it is overinflated, or if the catheter is reused. Sterile technique during insertion and manipulation of the catheter will minimize the risk of infection.

Despite its shortcomings and complications, the flotation pulmonary artery catheter is a safe and practical clinical device that provides reasonably accurate information about left ventricular function.

PETER L. TUXEN, MD

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## Anesthetic Management for Patients with Coronary Artery Disease

THE MOST CHALLENGING and important problem facing an anesthesiologist caring for a patient with coronary artery disease is the control of factors affecting myocardial oxygen supply and demand.

The major controllable determinant of myocardial oxygen supply is the diastolic arterial blood pressure. Below a critical lower pressure limit, reduced flow across narrowed coronary arteries may cause a loss of autoregulation of coronary flow. Therefore, anesthesiologists must keep patients' diastolic blood pressure high enough to ensure adequate coronary blood flow. Although critical lower pressure limits vary from patient to patient, most anesthesiologists attempt to maintain the diastolic pressure above 50 mm of mercury. One should suspect a problem with low myocardial oxygen supply if changes in ST segments, arrhythmias or pump failure occur.

The four major determinants of myocardial oxygen demand are heart rate, afterload (the systolic blood pressure plus any aortic valve pressure gradient), preload and the state of myocardial contractility. The rate-pressure product (heart rate times systolic blood pressure) correlates well with myocardial oxygen demand. In the presence

of an increasing rate-pressure product, changes in ST segments, arrhythmias or pump failure should lead an anesthetist to suspect that myocardial oxygen demand exceeds supply. In general, high myocardial oxygen demand is produced by patient anxiety, light anesthesia (especially during intubation), lack of  $\beta$ -adrenergic blockade, or the overuse of pressor or cardiotoxic drugs.

Adequate monitoring of patients with coronary heart disease requires a continuous electrocardiogram on which ischemic ST-segment changes can be seen. If possible, lead V5 should be monitored, since it is usually the most sensitive in evaluating changes in the ST-segment. In the absence of V lead capabilities, lead 2 is the best second choice. Direct intraarterial blood pressure monitoring is recommended so that second-to-second changes can be observed, and in addition, arterial blood-gas determinations should be made. If the disease is severe or there is substantial left ventricular dysfunction (or both), a pulmonary artery catheter (Swan-Ganz) should be placed in order to continuously determine the left ventricular filling pressures (preload).

It is important to realize that there are two major categories of patients with coronary artery disease, requiring notably different management techniques. The first includes those patients with little or no myocardial damage, a normal cardiac reserve, no history of cardiac failure, a left ventricular end-diastolic pressure (LVEDP) under 14 mm of mercury, ejection fraction over 0.4 and no left ventricular aneurysm. These patients tend to have hyperdynamic responses to any stimulus (for example, anxiety or pain). Management should include adequate preoperative discussion and sedation, adequate anesthesia,  $\beta$ -adrenergic blockade both preoperatively and intraoperatively, the use of intravenously given nitroglycerine or sodium nitroprusside (or both), intra-tracheal spray with lidocaine before tracheal intubation and possibly, metocurine instead of pancuronium. The use of enflurane or halothane, either as the major anesthetic agent, or as an adjunct to a narcotic technique, is desirable. All of the above measures tend to minimize increases in myocardial oxygen demand.

The second patient category includes patients with previous major cardiac damage, including ventricular aneurysm, mitral or aortic valvular dysfunction, heart failure or previous myocardial infarction, an LVEDP over 14 mm of mercury, an